MALIGNANT TRICUSPID ENDOCARDITIS: WITH A REPORT OF FIVE CASES.

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Right-sided endocarditis (fetal endocarditis) is familiar to all. Vegetations on the tricuspid or pulmonary valves in association with similar lesions on the valves of the left side of the heart are not rare, as autopsy statistics certify. But primary acute endocarditis confined to one or another of the valves of the right heart is infrequent.

Osler, in his Gulstonian Lectures, analyzed 209 cases of endocarditis and found the right heart alone involved nine times only. He mentions it as a rare finding and still more rarely diagnosed. Among 21,000 autopsies at Guy's Hospital, endocarditis confined to the tricuspid valve was encountered twelve times. At Bellevue Hospital among a series of 6800 autopsies primary tricuspid endocarditis was met with four times.

References to the lesion in text-books of medicine and even in monographs are scant indeed. Mackenzie dismisses it by simply mentioning its rarity. Herz does likewise. Hirschfelder calls attention to the rarity of organic tricuspid insufficiency in contrast to the frequent finding of relative insufficiency. The latter, he asserts, is probably present in every dying or failing heart. He calls attention to the fact that primary tricuspid endocarditis is usually a malignant endocarditis, a statement which is confirmed by other statistics. An unfavorable prognosis, therefore, usually follows its diagnosis. According to Broadbent the relative tricuspid insufficiency is usually not accompanied by a murmur unless the insufficiency is extremely marked. Strümpell refers to the lesion as follows: "Endocarditis on the tricuspid valve is seldom seen except as a secondary affection in old cases of heart disease. In a case of acute ulceration in a grown man seen by us the process was confined exclusively to the tricuspid valve and there were very many embolic abscesses in the lungs. This may be considered a great rarity."

The diagnostic problem which the condition presents is not without interest. Its rarity and obscure symptomatology on the one hand and the differentiation from the often present relative tricuspid insufficiency on the other make diagnosis oftentimes impossible. It is the intention of this paper to consider certain points in the diagnosis of this lesion. A more extended discussion on the diagnostic problem of tricuspid insufficiency is given in Young and Cotter's paper.

The percussion of the right heart and auscultatory symptoms of the tricuspid area do not aid us, chiefly because of the signs given by mitral disease and relative tricuspid insufficiency. However, the associated phenomena in other structures are of sufficient importance, at least to the extent that if found we shall be placed on our guard and suggest the possibility of this lesion. Of these the jugular pulse is most often present; less frequently a liver pulse, though in Young and Cotter's paper the order was reversed. But to distinguish between a jugular pulse of organic origin and one due to relative tricuspid insufficiency is impossible except occasionally during therapy. Relative tricuspid insufficiency is due to poorly contracting heart muscle. If upon the administration of digitalis the heart muscle improves the physical signs of relative tricuspid insufficiency (loss of pulsations, loss of congestion, loss of murmurs and diminution in area of cardiac dulness) become less marked or disappear entirely. The danger of mistaking such improvement for healed valve disease must not be overlooked. The reverse obtains in organic disease. With improvement in the muscular contractions the physical signs become more marked (due to a larger volume of blood being thrown back). The jugular pulse, due to tricuspid insufficiency, is a systolic pulse, and while not absolutely diagnostic is usually present only in tricuspid incompetency. Like all venous pulses it is produced by a slowly distending vessel. During the systole of the right auricle when the latter is emptying, systolic emptying of the jugular does not occur because of the back flow of the blood, and hence the vein remains distended in systole, in contradistinction to the normal jugular pulse and that due to congestion, as in mitral disease, for example, in which circumstance the jugular vein does empty itself, producing a negative pulse during systole. The sign, therefore, in tricuspid insufficiency—either relative or organic—is a jugular systolic (positive) pulse.

The hepatic venous pulse must not be confounded with aortic impulse transmitted to the liver. The latter is a short, very quickly appearing and disappearing impulse. The venous pulse in the liver is slow, markedly retarded and always in two phases, the time relationship being presystolic-systolic. It can usually be felt best when the abdominal muscles are relaxed and deep palpation is possible.

Manges described an interesting case of double mitral disease accompanied by tricuspid regurgitation, in which both the liver and the spleen pulsated. He could not account for the mechanism producing this phenomenon.

Lung infarction, with or without abscess formation, is almost constantly present and generally leads to a diagnosis of broncho-pneumonia.

Long-continued physical signs in the lung which are evanescent or rapidly changing, together with the heart signs and associated

jugular and hepatic pulse, should make us fairly certain of tricuspid endocarditis.

The following five cases of primary acute tricuspid endocarditis presented several interesting points clinically and at autopsy. The first four cases represent the Bellevue Hospital series; the fifth case was encountered among 429 autopsies in the American Army.

CASE I.—F. W., aged thirty-six years, admitted to Bellevue Hospital complaining of chills and fever for three weeks previous. On admission the temperature was 104° F., respiration 28, pulse 120. There was dulness and diminished breathing at the right base posteriorly. Tentative diagnosis: Pneumonia. The patient remained in the hospital for twenty-five days, during which time he ran a septic temperature, with rapid breathing and pulse averaging 120. The sputum was at times profuse and blood-tinged. Physical signs of the heart showed a systolic murmur about 2.5 cm. to the right of the apex, which was in the fifth space. No venous or hepatic pulsations noted.

At autopsy each pleural cavity contained about 400 c.c. of serofibrinous fluid. The pleural surfaces of both lungs were covered with a fine fibrin deposit, underneath which there were many large and small petechiæ. The left lung on section presented a glazed surface in the upper lobe, with slight increase in fibrous tissue. At the apex there were several calcareous tubercles. The lower lobe presented a dark reddish surface from which considerable blood exuded. The right lung presented a similar picture to the left except that there was a large wedge-shaped, dark reddish, sharply circumscribed area in the lower lobe. The bronchi on both sides were congested and contained about 30 c.c. of blood-stained serum.

The heart was of normal size and its surface showed a few petechiæ. The tricuspid ring admitted four fingers. The middle leaflet of the valve was the location of a large, cauliflower-like vegetation which extended down the chordæ tendineæ to the papillary muscles. The remaining valve segment showed old thickenings and scarrings. The remaining valves of the heart were entirely normal. The foramen ovale was patent, the right ventricular cavity somewhat dilated and the heart muscle pale brown in color. The coronary vessels are normal. The aorta showed a few small atheromata at its commencement.

The surface of the liver was smooth. On section it showed marked fatty infiltration.

The spleen was large, soft and grumous in appearance.

The remaining organs showed no changes worthy of note.

Anatomical diagnosis: Acute tricuspid endocarditis; patent foramen ovale; double serofibrinous pleurisy; hemorrhagic infarction of right lung; purulent bronchitis; healed apical tuberculosis; fatty infiltration of liver; acute splenic tumor; septicemia.

CASE II.—S. F., aged twelve years, admitted to Bellevue Hospital January 28, 1906, died February 12, 1906. Patient had had high fever and was delirious for one week previous to admission. General examination on admission showed a poorly nourished body; the skin and mucous membranes were pale; a few petechiæ were seen in the conjunctivæ and over the chest. The temperature was 104.2° F., respiration 36, pulse 124. Scattered bronchial breathing was noted over both lungs. This continued with occasional areas of dulness to flatness until death. The heart was slightly enlarged to the left by percussion and a loud, blowing systolic murmur was heard at the apex. Distinct pulsation was noted in both jugulars. There was little or no sputum. The temperature remained high, the pulse continued rapid and respirations became more labored. The patient developed a swelling over the right shoulder and right hip-joint, which was painful and tender, but showed no pus on aspiration. Blood culture showed the Staphylococcus pyogenes aureus. The white blood count was 18,200; polymorphonuclears 86 per cent.; the urine was full of albumin and casts. Diagnosis: Malignant endocarditis-septicemia.

At autopsy the pericardium was covered with a thick, shaggy film and contained about 100 c.c. of thin, purulent fluid. The heart was somewhat large and the muscle was pale and flabby. The endocardium was smooth. The right leaf of the tricuspid valve showed an area of thickening about 5 mm. in diameter, beneath which the valve was adherent to the right ventricle. Along the margin of this leaf there were many small verrucous vegetations and one large pendulous, reddish-gray, thrombotic mass which projected into the ventricular cavity. The remaining leaves of this valve were normal. The remaining heart valves were normal. The coronaries showed no lesions. The aorta was normal.

The lungs on the surface showed a number of sharply circumscribed, depressed and purplish areas which, on section, corresponded to hemorrhagic infarcts. In the center of several of these there were one or more minute yellowish foci. The parenchyma of the lung in the lower lobes showed considerable congestion and edema. The bronchi were slightly injected.

The spleen was markedly enlarged. On section it was dull reddish in color and fleshy in appearance.

Both kidneys on section showed numerous miliary abscesses on the cortex and in the substance, each surrounded by a prominent hemorrhagic zone.

The right tonsil on section showed a yellowish, purulent focus about 5 to 10 mm. in diameter.

Anatomical diagnosis: Purulent pericarditis; acute tricuspid endocarditis; miliary abscesses of lungs and kidneys; hemorrhagic infarction of lung.

Bacteriology: Staphylococcus pyogenes aureus obtained from the spleen.

CASE III.—E. M., white, aged sixty-eight years, was admitted to Bellevue Hospital on July 14, 1910, with a history of swelling of the abdomen and extremities, chills and fever of several weeks' duration. Examination revealed an elderly, emaciated female, with moderate edema of the lower extremities. The heart was not enlarged by percussion, the sounds being faint but clear. The lungs showed bronchovesicular breathing in both upper lobes, with many fine rales scattered through both organs. The temperature was 102° F., pulse 120, respiration 24. The abdomen was prominent and there was an umbilical hernia which was ulcerated and through which a hard mass could be felt. There was no tenderness or rigidity. The abdomen was flaccid; no fluid was found. During her stay in the hospital the patient became progressively worse, the signs in the lungs spread, the heart became irregular and a systolic murmur developed over the tricuspid area. Urine: Acid; sp. g., 1.012; trace of albumin and granular casts. Patient died on July 30, 1910. Diagnosis: Ovarian tumor; nephritis; pneumonia.

Autopsy. The body was that of a very much emaciated white female. The skin was pale and there was no edema of the subcutaneous tissues. There was a large umbilical hernia, the mass protruding for a distance of from 8 to 10 cm., the apex being ulcerated and exuding fluid on pressure. The abdominal wall was considerably thickened by dense connective tissue to which a large tumor mass was adherent, occupying the entire anterior and lateral portions of the abdominal cavity. On dissection this mass was found to be a large multilocular cystadenoma attached to the right broad ligament. A diffuse serofibrinous exudate was present.

Both lungs showed cicatrices and areas of interstitial pneumonia at their apices. The left lower lobe was reddish in color, firm and smooth on section. The branches of the pulmonary artery of the left lung were the seat of reddish, soft thrombi which were firmly adherent in places. The right lung was likewise the seat of thrombi and small areas of lobular pneumonia. The bronchi were extremely congested and partially filled with mucopurulent exudate. The longitudinal striations were prominent. The upper lobes were pale and inelastic, the lower lobes dark, congested and edematous.

The pericardial sac contained about 100 c.c. of clear fluid. The heart was small and the vessels tortuous. The tricuspid valve showed a vegetative growth about 8 mm. in length, with small ulcerative areas surrounding it. The remaining heart valves were normal. The heart muscle was pale and very flabby, with some prominent whitish delineations.

The kidneys were small and contracted, the cortex normal, pyramids indistinct and the surface granular.

The remainder of the autopsy was irrelevant.

Anatomical diagnosis: Multilocular cystadenoma of the right ovary; acute tricuspid endocarditis; chronic interstitial pneumo-

nitis; lobular pneumonia; emphysema; chronic bronchitis; chronic interstitial nephritis.

CASE IV.—W. S., aged fifty years, laborer, was admitted to Bellevue Hospital on October 14, 1914. The wife of the patient stated that he had caught cold five days previous to admission and that fever and cough steadily increased. Physical examination revealed a moderately developed and poorly nourished adult male, acutely ill. The right eye was entirely destroyed; the left pupil was regular and reacted to light and accommodation; no ptosis. The pulse was regular and of good force and volume. The vessel walls felt thickened and tortuous. Blood-pressure, 135. The apex-beat was heard in the sixth left interspace, four inches from the midsternal line. Pulsation was also felt in the fifth space in palpable thrills. The heart sounds were clear and of fair muscular quality; no murmurs heard; pulmonic second sound accentuated; right border of heart not obtained. Respirations were labored and expansion of the right chest diminished. In the left chest the percussion note was resonant. The breath sounds were bronchovesicular. Numerous coarse bubbling rales were heard laterally and over the lower lobe, chiefly at the base. Fremitus was increased at about the angle of the capsula. Right chest: Down to the sixth rib there was flatness; bronchial breathing with increased fremitus; many fine and coarse rales were present. Below this the note was resonant and the breath sounds bronchovesicular. Posteriorly, from apex to base, there was flatness, increased fremitus and bronchial breathing, with many fine and coarse rales. No pleural friction heard.

The abdomen was markedly distended and tympanitic; there were no palpable masses and no rigidity or tenderness. The liver was palpable two fingers' breadth below the free border of the ribs in the nipple line.

The patient perspired freely. There was a scar on the left side of the face and scalp and numerous petechiæ over the body. There was no edema or swelling. The face, ears and extremities had a cyanotic tinge.

Bones and Muscles. Limitation of extension at both elbows; slight irregularity in bone at right ankle.

Head. Rachitic; furrow between frontal bases.

Provisional Diagnosis. Lobar pneumonia (right).

On October 15 the patient was considerably worse. Cyanosis and dyspneic respirations more marked; perspiration very profuse. Died at 4.40 P.M. on October 15, 1914.

Urine. Acid reaction; albumin; granular casts; sp. g., 1.018.

Temperature, 102.8° F. on admission; remained at 102° until death. Respiration, 36 to 48. Pulse, 120 to 144.

Blood culture was positive for pneumococcus. Wassermann reaction four plus.

Autopsy. The body was that of an elderly white male, 160 cm.

in length, of fair nutrition and well-developed musculature. The right eye was absent. There were numbers of petechiæ distributed over the body.

The lungs were firmly adherent by dense fibrous adhesions. The right lung was the seat of a massive pneumonia—the upper portion in the stage of gray hepatization, the lower in the red hepatized stage. The left lung was very edematous and showed a small area of consolidation (about 4 cm. in diameter), which was somewhat soft and necrotic. The bronchi were intensely congested.

The heart was large. The right auricle was markedly distended by a blood-clot. The tricuspid valve was considerably thickened and showed a few minute verrucous vegetations on its auricular surface, averaging about 1 mm. in diameter. The pectinate muscles in the right ventricle were prominent and the papillary muscles were hypertrophied. The wall of the right ventricle was about 5 mm. in width. The remaining valves were normal, as were also the coronaries and aorta. The left ventricle was hypertrophied.

The spleen was small, friable and grayish-red in color. The kidneys were grayish red in color, the cortex swollen and the markings indistinct.

Anatomical diagnosis: Lobar pneumonia (right); bronchopneumonia (left); pulmonary edema; acute bronchitis; acute tricuspid endocarditis; chronic valvulitis (tricuspid); chronic adhesive pleuritis; septicemia.

This case in all probability represents vegetations developing on a weakened valve, the seat of a former rheumatic process.

Case V.—O. C., male, mulatto, aged twenty-five years, was admitted to Bellevue Hospital on November 28, 1918, complaining of fever and pains in the chest. His temperature was 104°, pulse 120, respiration 30. There was scattered dulness and bronchial breathing over the right lower and middle lobes and over the left lower lobe. A diagnosis of bilateral lobar pneumonia was made. The patient stated that he had had a "cold" for two weeks previous to admission, with chills and fever, and had suffered from rheumatism and tonsillitis frequently for six or seven years past.

The signs in the chest continued until December 10, when they became less marked, though an area of bronchial breathing was noted in the right axilla. During this time the patient expectorated profusely, at first blood-tinged, later purulent. Toward the middle of the month the chest was almost clear, but on December 30 there were small areas of dulness and bronchial breathing throughout both lungs. The heart was negative. These signs continued, with varying intensity, until January 28, when 300 c.c. of thin, yellowish, purulent fluid were aspirated from the right chest. On February 1 the patient was operated on for empyema. He did not improve after the operation and died on February 4.

During the entire illness the patient had a typically septic tem-

perature, ranging from 100° to 105.4° F. His pulse was rapid, averaging 120 to 130; respirations were likewise always rapid. He was very restless, often irritable, and lost a good deal of weight. His sputum was profuse and frequent attacks of coughing annoyed him very much. He was delirious at times. Of the laboratory examinations, unfortunately, it was possible to find only a urine report which showed albumin, casts, pus and epithelium. No blood cultures were taken, neither was the empyema fluid cultured. The clinical diagnosis was lobular pneumonia and empyema.

Autopsy. The body was that of a slender adult mulatto, 179.5 cm. in length, poorly nourished. The external configuration and development of the body and its appendages were normal. Rigor mortis was marked. There was an incised wound 1.5 cm. long in the right posterior axillary line, in the seventh interspace and a larger but similar wound in the tenth interspace in the back. The distribution of hair was abundant, coarse, black, short and kinky. There were a few superficial scars over both tibiæ. The conjunctivæ were pale; the scleræ clear; the pupils equal, regular and contracted; no petechiæ. The ears and nose externally were normal. The lips were excoriated, the teeth in fair condition, the tongue dry, furrowed and covered with a brownish coating on the surface. There were old operative scars over both inguinal regions.

The thymus was replaced by fat. The ribs were normal. Many firm and several loose fibrous adhesions on the right side formed numerous encapsulated sacs which contained yellowish purulent fluid. In the left chest there were about 200 c.c. of cloudy fluid which contained fibrin flakes. A few easily destroyed adhesions were present at the apex, but the base was firmly adherent to the diaphragm.

The precordial area measured 12 cm. across. The pericardial sac contained 5 to 10 c.c. of clear, straw-colored fluid. The heart was large, especially the right side, weighing about 450 gm. A few petechiæ were present in the posterior epicardium. The muscle was soft and flabby and pale, yellowish brown in color, measuring 18 mm. in width at the base of the right ventricle. The tricuspid valve was covered almost everywhere by numerous verrucous vegetations, especially on the posterior cusp where the vegetations had pyramided with its apex directed downward, hanging free in the cavity of the ventricle. There were a few minute verrucæ on several of the chordæ tendineæ. The remaining valves were perfectly normal. The fossa ovalis was without a foramen. The coronary vessels were not opened but their commencement was normal.

The left lung was covered with a thin purulent fibrin, and there were many subpleural hemorrhages. The organ was rather firmly adherent at the base. The right lung was covered with a shaggy fibrinous exudate. On section both lungs presented an irregularly firm, reddish-gray, mottled surface, exuding frothy fluid in many places. In about a dozen places in each lung there were firm, sharply

circumscribed, dark hemorrhagic areas, many of them pyramidal in shape and varying in size from 1 or 2 cm. to 4 or 5 cm. At the base of the right lung posteriorly and near the surface there was an area about 3 cm. in diameter, soft and necrotic, presenting multiple miliary abscesses which contained yellowish purulent fluid. The bronchi were intensely congested. The lymph nodes were anthracotic, large and succulent.

The spleen was large, weighing 600 gm. It was soft and, on section, presented a dull red, fleshy and grumous surface in which the lymphoid structures were not recognizable.

The right kidney on section presented near its lower pole an encapsulated area, about 2.5 cm. in diameter, containing some opalescent fluid. In the pelvis of this kidney there was a large irregular, tri-branched brownstone and several smaller stony granules. The pelvis and upper part of the right ureter were moderately injected. On both kidneys the capsule bulged but stripped off readily. The cortex was red and swollen, the pyramids prominently delineated. The ureter on the left side was normal.

The remainder of the autopsy was irrelevant.

Anatomical diagnosis: Acute tricuspid endocarditis; cardiac hypertropby; fatty infiltration of myocardium; multiple pulmonary infarcts; lobular pneumonia; pulmonary edema and congestion; acute bronchitis; empyema; adbesive pleuritis (left base); hyperplastic splenitis; renal calculus; pyonephrosis; acute parenchymatous degeneration of kidneys; septicemia; operative wounds of thorax.

Bacteriology. Cultures were made from the heart valve and from a lung infarct, and both sbowed Streptococcus hemolyticus in pure culture.

Summary. 1. Five cases of tricuspid endocarditis are herewitb reported.

2. The lesion is rare.

3. Bacteriologically, the lesion is associated with different varieties of actively pathogenic microörganisms.

4. Lung emboli or abscesses are practically invariably present.

5. The prognosis is extremely unfavorable, the average duration of life from onset of symptoms being two to three months.

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